



HUMAN AKT-3

FIELD OF THE INVENTION

- 5 The present invention is concerned with cloning and expression of a new human serine/threonine kinase termed "Akt-3" and, in particular, with nucleic acid molecules encoding the Akt-3 protein, the protein itself and compounds which can be used to inhibit cell survival.

BACKGROUND OF THE INVENTION

- 15 A characteristic feature of many cancer cells is their ability to grow independently of adhesion. In contrast, when untransformed endothelial cells are prevented from adhering to the extracellular matrix (ECM), they undergo apoptosis (Frisch & Francis, 1994; Meredith et al, 1993). The process by which normally
- 20 adherent cells are triggered to undergo apoptosis when they are unable to adhere to ECM has been termed "anoikis" (Frisch & Ruoslahti, 1997) and is an example of the effect on a cell of removal of a survival factor. Changes in signalling by adhesion molecules
- 25 can lead to resistance to anoikis (Frisch & Ruoslahti, 1997) and this may contribute to the mechanism whereby cancer cells that grow independently of adhesion are able to avoid anoikis.
- 30 Akt (also known as protein kinase B (PKB) or "related to A and C protein kinase" (RAC-PK)) is a serine/threonine kinase that has been implicated in regulating cell survival (Khwaja et al., 1997; Dudek et al., 1997; Kauffmann-Zeh et al., 1997; Kennedy et al., 1997; Datta et al., 1997; Marte & Downward, 1997). Akt can inhibit apoptosis induced by detachment from ECM (Khwaja et al., 1997), as well as by survival factor withdrawal (Kennedy et al., 1997; Ahmed et al., 1997; Dudek et al., 1997; Kauffman-Zeh

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Substitute
specification
not entered
SDP
5/27/05

THE UNIVERSITY OF CHICAGO